The Effect of Diarrheal Disease on the Absorption and utilization of Vitamin A, an annotated bibliography. By

Donna Nager

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THE EFFECT OF DIARRHEAL DISEASE ON THE ABSORPTION AND UTILIZATION OF VITAMIN A

AN ANNOTATED BIBLIOGRAPHY

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THE EFFECT OF DIARRHEAL DISEASE ON THE ABSORPTION AND UTILIZATION OF VITAMIN A

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Lala, V.R. and Reddy, V.: Absorption of B-Carotene from green leafy vegetables in undernourished children. Am. J. Clin. Nutr. 23:110, 1970.

In this study investigations were undertaken to obtain the following information: a) carotene-balance studies in preschool children and b) determination of serum vitamin A levels before and after feeding green leafy vegetables as the sole source of vitamin A.

It is of interest that in their discussion the authors note that in two children who had infection, absorption of carotene was quite low while in the undernourished children without infection absorption of carotene was as efficient as in normal adults. This finding is in line with the observations reported by Heymann (Am J Diseases Children 51: 273, 1936) that infection decreases the absorption of carotene.

Nalin, D.R., Russell, R. and Levine, M.M.: Reduced vitamin A (vA) absorption after enteric infections. Proceedings of the 11th International Congress of Chemotherapy - 19th Interscience Conference on Antimicrobial Agents and Chemotherapy, 1979.

In this study the authors tested the hypothesis that vitamin A malabsorption induced by diarrhea attacks might be significantly interfering with the efficacy of vitamin A prophylaxis, since high prevalence of vitamin A deficiency, which remains an important cause of blindness among children in the developing countries, typically occurs in areas where diarrheal diseases are endemic.

Studies of vitamin A tolerance were performed before and after induced diarrhea in free-living volunteers. Control tests were performed on normal non-infected volunteers and on volunteers infected with nonpathogenic bacteria. Tolerance tests were performed by measuring blood vitamin A before and after an oral vitamin A dose (350,000 I.U.).

Normal noninfected control volunteers, and those infected with control nonpathogenic organisms, developed a rise in peak serum vitamin A when the test was repeated within a one-week period.

Peak serum vitamin A declined following diarrheal episodes caused by enterotoxigenic Escherichia coli, parvoviruses and cholera. Infection with certain strains of Escherichia coli, as well as parvoviruses, decreased vitamin A tolerance even in the absence of diarrhea or other symptoms.

The authors concluded that certain intestinal infections, viral and bacterial, can decrease vitamin A tolerance, suggesting interference with intestinal absorption of vitamin A. The decrease can occur in asymptomatic as well as symptomatic individuals. As these pathogens are known to be prevalent in developing countries, it appears likely that they are an important cause of vitamin A malabsorption.

They recommend that vitamin A prophylaxis should be linked with enteric disease control programs in order to more effectively reduce blindness due to vitamin A deficiency.

Oomen, H.A.P.C., McLaren, D.S. and Escapini, H.: A global survey on xerophthalmia. Trop. and Geogr. Med. 16:271, 1964.

Diarrhoea. The case of xerophthalmia which in a purist sense is only caused by lack of vitamin A must be an extreme rarity. The average case, especially of keratomalacia, always has a story of diarrhoea, fever or ascariasis. It is the "diarrhoea months" which produce the cases in Japan, Korea, Near East, North Africa etc.: where the climatic variations are not so great it is just diarrhoea. If the diarrhoea is epidemic, as that caused by Shigella, such a wave of dysentery may be followed by a smaller wave of xerophthalmia. Amoebic dysentery sometimes has an especially bad reputation. But the first connection which busy practitioners make with xerophthalmia is invariably diarrhoea.

Admissions To Children's Ward (±150 beds) 1958-1959 of children from 1-5 years, Central General Hospital, Semerang (Java)

Diagnosis	Total Admissions	with symptoms of xerophthalmia	
		number	Percent
Malnutrition	461	165	36
Amoebic dysentery[1]	40	14	35
Bronchopneumonia/ gastroenteritis	76	9	12
Measles[1]	67	7	10
Gastroenteritis/ intoxication	581	44	8
Pulmonary tuberculosis	124	8	6
Typhoid	34	1	3
Pneumonia (acute)	61	1	2
Diphtheria	113	1	1
Total Admissions	5699	332	6

[1] Only serious or complicated cases were admitted

Rosenberg, I.H., Solomons, N.W. and Schneider, R.E.: Malabsorption associated with diarrhea and intestinal infections. Am. J. Clin. Nutr. 30:1248-1253, 1977.

An episode of diarrhea causes weight loss and a temporary cessation of growth in infants and children. Diarrhea is accompanied by malabsorption of sugars, nitrogen, fats and micronutrients:

Nutritent malabsorption in acute diarrheal disease

Nutrient

- Sugar
  - Monosaccharides (glucose, xylose)
  - Disaccharides (lactose)
- Nitrogen, protein, amino acids
- Fat
- Vitamins
  - B_{12}
  - Folate
  - Vitamin A
- Minerals and trace elements

The mechanisms by which acute diarrheal disease produce malabsorption have not been studied carefully:

Malabsorption in diarrheal disease: pathogenetic factors

Framework of possible mechanisms

A. Intraluminal events
  1. Bacterial overgrowth
    a. competition
    b. metabolism (fermentation)
    c. gas
    d. short chain fatty acids
    e. bile acids
  2. Osmotic effects
B. Cellular events
  1. Pharmacotoxic
  2. Cytotoxic
C. Villous abnormalities

The severity of the nutritional deficit is related to the duration and magnitude of the malabsorption in each individual situation. The nutritional costs of malabsorption may pose a major threat if diarrhea becomes chronic or recurrent.

Sivakuma, B. and Reddy, V.: Absorption of labelled vitamin A in children during infection. Br. J. Nutr. 27:299, 1972.

The effects of infection on the metabolism of vitamin A in children were investigated.

The intestinal absorption of $(11,12-^{3}H_{2})$ retinyl acetate was studied in five apparently normal children, eight children with respiratory infection and three with gastroenteritis. Their ages ranged from 2 to 10 years.

The absorption of vitamin A was significantly lower in children with respiratory infection or gastroenteritis than in normal children.

The mean level of absorption in the control group was 99.2%. Two children who had seven to eight loose stools/day showed 64.4% and 64.8% absorption and the other subject, who had four to five loose stools/day had an abosrption of 81.4% of the label administered. The authors state that the defective absorption in these children could be due either to infection or to the excessively rapid passage of the intestinal contents, or to both.

It was concluded that repeated attacks of respiratory infections and diarrhea may enhance the vitamin requirements by interfering with intestinal absorption of the vitamin. In children whose dietary intakes are low and whose body stores of the vitamin are marginal, this may precipitate clinical manifestations of vitamin A deficiency. In addition to an inadequate dietary intake of vitamin A, a high incidence of repeated attacks of infection may contribute significantly to the wide prevalence of vitamin A deficiency in children in poor communities.

The authors also noted that the mechanism by which the absorption of vitamin A is depressed needs study.

Mahalanabis, D., Jalan, K.N., Maitra, T.K. and Agarwal, S.K.: Vitamin A absorption in ascariasis. Am. J. Clin. Nutr. 29:1372-1375, 1976.

Vitamin A absorption was studied in a group of 28 adult patients with ascariasis and 12 healthy adult controls, using a simplified vitamin A absorption test. In over 70% of the patients with ascariasis malabsorption of vitamin A was demonstrated. Stool egg counts for ascaris were not related to the degree of vitamin A malabsorption. Of the 23 patients in whom a D-xylose absorption test was performed, seven showed excretion less than 20% in 5 hours. Immediately after expulsion of the worms, vitamin A absorption improved in 13 out of 14 patients tested (in nine to normal level). The results of this study suggest that ascariasis in populations on marginal intakes of vitamin A and its precursors is an important contributing factor in producing clinical vitamin A deficiency. Authors summary.

Mahalanabis, D., Simpson, T.W., Chakraborty, M.L., Ganguli, C., Bhattacharjee, A.K. and Mukherjee, K.L.: Malabsorption of water miscible vitamin A in children with giardiasis and ascariasis. Am. J. Clin. Nutr. 32:313-318, 1979.

Vitamin A absorption was studied using a water-miscible oral preparation of vitamin A in 19 children ages 1½ to 9 years old with giardiasis and/or ascariasis, both before and after their eradication with appropriate therapy, and in three children without parasites. Marked impairment of vitamin A absorption was noted when administered in a water miscible form in children with 1) combined infection with Giardia lamblia and Ascaris lumbricoides, 2) giardiasis alone, and 3) in a proportion of children with ascariasis alone. In children with both giardiasis and ascariasis eradication of the infections promptly lead to a significant improvement in vitamin A absorption and restored it to normal. Children with giardiasis alone also showed improved vitamin A absorption after therapy. In children with ascariasis alone successful therapy did not lead to a statistically significant improvement. Authors summary.

Sivakumar, B. and Reddy, V.: Absorption of vitamin A in children with ascariasis. J. Trop. Med. Hyg. 78:114, 1975.

An investigation was undertaken by the authors to study the absorption of vitamin A in children with roundworm infestation, as it has been observed that children with ascariasis tend to have a high incidence of ocular signs of vitamin A deficiency.

Absorption of labelled vitamin A was studied in normal children and in children with roundworm infestation. Absorption of the vitamin was significantly lower in children with ascariasis. In two subjects, in whom the studies were repeated absorption of vitamin A was significantly increased after deworming. These results suggest that ascariasis may aggravate vitamin A deficiency by interfering with the absorption of the vitamin.

In their discussion the authors state that the mechanism by which the worms interfere with absorption of vitamin A is not clear. They say it is possible that the presence of worms may lead to an alteration in the structural integrity of the mucosa and in intestinal motility.

They conclude: the results of these studies suggest that infestation with ascariasis can aggravate vitamin A deficiency, particularly if the dietary intakes are marginal, by interfering with intestinal absorption; and, prevention and control of vitamin A deficiency through prophylactic programmes would have better impact if combined with periodic deworming of children of poor communities who harbour roundworms.

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